

3. (Amended) A method for treating a disorder characterized by neuronal cell loss, comprising administering to a mammal a composition comprising a molecule that overcomes morphogen inhibition, thereby to potentiate growth-promoting effects of endogenous morphogens.

4. (Amended) A method for treating a neurodegenerative disorder, comprising administering to a mammal a composition comprising a molecule that overcomes morphogen inhibition.

5. (Reiterated) The method of claim 1, wherein said morphogen activity is endogenous.
6. (Reiterated) The method of claim 1, wherein said morphogen activity is the result of an exogenously provided morphogen.
7. (Reiterated) The method of claim 4, wherein said composition further comprises a morphogen.

8. (Amended) The method of claim 3 or 4, wherein said disorder is Alzheimer's disease, Parkinson's disease, Huntington's disease, senile dementia, alcohol-induced dementia, or stroke.

9. (Amended) The method of claim 1, 2, 3 or 4, wherein said agent that overcomes morphogen inhibition is a cytokine antagonist, a retinoid antagonist, or a protein kinase A inhibitor.

10. (Reiterated) The method of claim 9, wherein said cytokine antagonist is a neuropoetic cytokine antagonist.

11. (Amended) The method of claim 10, wherein said neuropoetic cytokine antagonist is an LIF antagonist or a CTNF antagonist.

12. (Amended) The method of claim 11, wherein said LIF antagonist is a monoclonal antibody to the gp130 protein.

13. **(Reiterated)** The method of claim 9, wherein said retinoid antagonist is a retinoic acid receptor antagonist.
14. **(Reiterated)** The method of claim 9, wherein said retinoid antagonist is a retinoid X receptor antagonist.
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15. **(Amended)** The method of claim 9, wherein said protein kinase A inhibitor is (2-p-bromocinnamylaminoethyl)-isoquinolinesulfonamide, an enantiomer of dibutyryl cAMP, or an enantiomer of cAMP.
16. **(Amended)** The method of claim 7, wherein said morphogen comprises an amino acid sequence selected from a sequence: (a) having at least 70% homology with the C-terminal seven-cysteine skeleton of human OP-1, residues 330-431 of SEQ ID NO: 2; (b) having greater than 60% amino acid sequence identity with said C-terminal seven-cysteine skeleton of human OP-1; (c) defined by Generic Sequence 7, SEQ ID NO: 4; (d) defined by Generic Sequence 8, SEQ ID NO: 5; (e) defined by Generic Sequence 9, SEQ ID NO: 6; (f) defined by Generic Sequence 10, SEQ ID NO: 7; or (g) defined by OPX, SEQ ID NO: 3.
17. **(Amended)** The method of claim 7, wherein said morphogen is human OP-1, mouse OP-1, human OP-2, mouse OP-2, 60A, GDF-1, BNT2A, BMT2B, DPP, Vgl, Vgr-1, BNW3, BNW5, or BW6.
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18. **(Reiterated)** The method of claim 7, wherein said morphogen is OP-1.
19. **(Amended)** The method of claim 1, wherein the molecule binds an endogenous ligand for a cytokine receptor or a retinoid receptor.
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20. **(Reiterated)** The method of claim 19, wherein said cytokine receptor is a neuropoietic cytokine receptor.
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21. **(Amended)** The method of claim 20, wherein said neuropoietic cytokine receptor is an LIF receptor or a CTNF receptor.

22. **(Reiterated)** The method of claim 19, wherein said retinoid receptor is a retinoic acid receptor.

23. **(Reiterated)** The method of claim 19, wherein said retinoid receptor is a retinoid X receptor.

24. **(Amended)** The method of claim 1, wherein the molecule is a cAMP-dependent messenger pathway inhibitor.

25. **(Amended)** The method of claim 24, wherein said cAMP-dependent messenger pathway inhibitor comprises a protein kinase A inhibitor.

26. **(Amended)** The method of claim 25, wherein said protein kinase A inhibitor is (2-p-bromocinnamylaminoethyl)-isoquinolinesulfonamide, an enantiomer of dibutyryl cAMP, or an enantiomer of cAMP.

a7 27. **(Amended)** A screening method for identifying a molecule that potentiates morphogen activity, comprising (1) providing a test cell comprising a morphogen inhibitory element, wherein said test cell, when contacted with OP-1, does not undergo tissue morphogenesis; (2) exposing said test cell to OP-1 and a candidate molecule; and (3) identifying a molecule that potentiates morphogen activity as a candidate that overcomes morphogen inhibition and permits said test cell to undergo OP-1-induced tissue morphogenesis.

28. **(Amended)** The screening method of claim 27, wherein said test cell is obtained from: sympathetic nerves, hippocampus, cerebral cortex, striatum, kidney, liver, adrenals, urinary bladder, or testes.

29. **(Reiterated)** A molecule identified by the method of claim 27.

30. **(Reiterated)** The molecule of claim 29, wherein said molecule is a protein.

31. **(Reiterated)** The molecule of claim 29, wherein said molecule is an inorganic molecule.

32. **(Reiterated)** The molecule of claim 29, wherein said molecule is an organic molecule.